WHAT IS CLAIMED IS:

1. A method for treating a condition involving cytokine-mediated toxicity comprising administering to an individual an effective amount of an agent capable of inhibiting MIF biologic activity.

2. The method of Claim 1 in which the agent is an MIF antagonist.

3. The method of Claim 2 in which the antagonist is an anti-MIF antibody.

4. The method of Claim 2 in which the antagonist is a soluble MIF-receptor.

5. The method of Claim 2 in which the antagonist is a small organic molecule.

6. A method for treating a condition involving cytokine-mediated toxicity comprising administering to an individual an effective amount of an agent capable of inhibiting MIF receptor biologic activity.

7. The method of Claim 6 in which the agent is an MIF-receptor antagonist.

8. The method of Claim 7 in which the antagonist is an anti-MIF-receptor antibody.

9. The method of Claim 7 in which the antagonist is a biologically inactive MIF analog which is capable of binding to MIF receptor.

10. The method of Claim 7 in which the antagonist is a small organic molecule.

11. A method for treating a condition involving cytokine-mediated toxicity comprising administering to an individual an effective amount of an agent capable of inhibiting MIF gene expression.

12. The method of Claim 11 in which the agent is an antisense molecule complementary to MIF mRNA.

13. The method of Claim 11 in which the agent is a ribozyme molecule specific for MIF mRNA.

14. The method of Claim 11 in which the agent is a triple helix component.

15. The method of Claim 11 in which the agent is a steroid.

16. A method for treating a condition involving cytokine-mediated toxicity comprising administering to an individual an effective amount of an agent capable of inhibiting MIF receptor gene expression.

17. The method of Claim 16 in which the agent is an antisense molecule complementary to MIF receptor mRNA.

18. The method of Claim 16 in which the agent is a ribozyme molecule specific for MIF receptor mRNA.

19. The method of Claim 16 in which the agent is a triple helix component.

20. A method for treating a condition involving cytokine-mediated toxicity comprising administering to an individual an effective amount of an agent that inhibits MIF release.

21. The method of Claim 20 in which the agent inhibits steroid induced release of macrophage MIF.

22. The method of Claim 21 in which the agent is administered in combination with a steroid.

23. The method of Claim 20 in which the agent inhibits toxin induced release of macrophage MIF.

24. The method of Claim 20 in which the agent inhibits TNFα-induced release of macrophage MIF.

25. The method of Claim 20 in which the agent inhibits IFN-γ induced release of macrophage MIF.

26. The method of Claim 20 in which the agent inhibits LPS induced release of pituitary MIF.

27. A method for treating a condition involving cytokine-mediated toxicity comprising administering to an individual an effective amount of (a) an agent capable of inhibiting MIF biologic activity, MIF receptor biologic activity, MIF gene expression, MIF receptor gene expression, or MIF release in combination with (b) anti-TNFα, anti-IL-1, anti-IFN-γ, IL-1RA, a steroid, a glucocorticoid, or IL-10.

28. The method of Claim 27 in which the agent is an MIF-antagonist.

29. The method of Claim 28 in which the antagonist is an anti-MIF antibody.

30. The method of Claim 28 in which the antagonist is a soluble MIF-receptor.

31. The method of Claim 28 in which the MIF-antagonist is administered in combination with a soluble receptor for TNFα, IL-1 or IFN-γ.

32. The method of Claim 28 in which the antagonist is a small organic molecule.

33. The method of Claim 1, 6, 11, 16, 20 or 27 in which the condition is septic shock.

34. The method of Claim 1, 6, 11, 16, 20 or 27 in which the condition is inflammation.

35. The method of Claim 1, 6, 11, 16, 20 or 27 in which the condition is autoimmunity.

36. The method of Claim 1, 6, 11, 16, 20 or 27 in which the condition is cachexia.

37. The method of Claim 1, 6, 11, 16, 20 or 27 in which the condition is a viral infection.

38. The method of Claim 1, 6, 11, 16, 20 or 27 in which the condition is a bacterial infection.

39. A method for inhibiting the toxic side effects of therapeutic steroids, comprising administering to an individual an effective amount of (a) an agent that modulates MIF biologic activity, MIF

receptor biologic activity, MIF gene expression, MIF receptor gene expression, or MIF release, and (b) the therapeutic steroid.

40. The method of Claim 39 in which the modulation is inhibition.

41. A method for enhancing the anti-inflammatory activity of therapeutic steroids, comprising administering an effective amount of (a) an agent that modulates MIF biologic activity, MIF receptor biologic activity, MIF gene expression, MIF receptor gene expression, or MIF release, and (b) the therapeutic steroid.

42. The method of Claim 41 in which the modulation is inhibition.

43. A method for detecting levels of MIF in a body fluid as an indicator of a disease condition comprising:

(a) contacting a test sample with an MIF binding partner under conditions and for a time sufficient to form MIF-binding partner complexes; and

(b) detecting the MIF-binding partner complexes.

44. The method of Claim 43 in which the MIF binding partner is an anti-MIF antibody.

45. A method for identifying compounds that inhibit cellular release of MIF, comprising:

(a) adding a test compound to a culture of cells that release MIF upon exposure to an MIF-inducer;

(b) adding the MIF-inducer to the cell culture;

(c) detecting any MIF released into the cell culture medium.

46. The method of Claim 45 in which the cells are a macrophage cell line.

47. The method of Claim 46 in which the MIF-inducer is a steroid.

48. The method of Claim 46 in which the MIF-inducer is a toxin.

49. The method of Claim 46 in which the MIF-inducer is TNFα.

50. The method of Claim 46 in which the MIF-inducer is IFN-α.

51. The method of Claim 45 in which the cells are a pituitary cell line.

52. The method of Claim 51 in which the MIF-inducer is a steroid.

53. The method of Claim 51 in which the MIF-inducer is a toxin.

54. An MIF receptor having an amino acid sequence comprising: Ile-X-His-Asn-Thr-Val-Ala-Thr-Glu-Ile-(Ser)-(Gly)-Tyr-Asn-(Asn/Gly)-(Ala)-(Met).

55. An MIF receptor having an amino acid sequence comprising: Ala-Lys-Lys-Gly-Ala-Val-Gly-Gly-Ile.

56. A cell line capable of expressing an exogenous MIF coding sequence.

57. A transgenic animal in which MIF gene expression is modified.

58. The transgenic animal of Claim 57 in which the modified MIF gene expression is inhibited.

59. A transgenic animal in which MIF receptor gene expression is modified.

60. The transgenic animal of Claim 59 in which the modified MIF receptor gene expression is inhibited.

61. An antibody that immunospecifically binds to MIF.

62. The antibody of Claim 61 which is a monoclonal antibody.

63. A method for augmenting an immune response to an antigen comprising administering to an individual an effective amount of MIF in combination with an antigen to induce an enhanced response to the antigen.

64. The method of Claim 63 in which the response is mediated by antibodies.

65. The method of Claim 63 in which the response is mediated by T cells.

Add 5 a2

Add C2

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